(source not stated), which ran a normal course, and terminated about the twenty-second day. During the attack the stomach-pump was used daily before breakfast, and the liquid removed carefully examined. In quantity it was less than previously, and scarcely reached 100 cubic centimetres. The main difference in its composition revealed by analysis was *the absence of free hydrochloric acid*, although its reaction was faintly acid, probably from the presence of lactic or acetic acid. A flake of fibrin remained quite undigested whilst in some of the filtered liquid in the cold; whereas the addition of an equal volume of 0.2 per cent. dilute hydrochloric acid caused its rapid disappearance. On the cessation of fever, on June 7, Dr. Von den Velden naturally expected that hydrochloric acid would again be found in the gastric liquid. He had, however, to wait until the *eighth* day after the temperature had become normal before traces of it reappeared. On the *ninth* day 0.08 per cent. was detected, and from this time onwards the percentage ranged between the limits mentioned above. It might be objected that since in this case the gastric contents were removed before food had been ingested, the absence of acid might depend on the absence of a stimulus to its secretion; but this objection fails, because the patient happened one morning to breakfast before being pumped, and still not a trace of free hydrochloric acid was present. The absence of acid was also not due to the patient's diet during the period of fever, for the latter consisted, *inter alia*, of milk, *bouillon* containing eggs, and other nitrogenized substances, in the presence of which acid juice would normally be secreted. Lastly, there was no increased secretion of alkaline mucus which would mask the free acid by neutralizing it as soon as secreted. This case confirms the observations of Pavy, Hoppe-Seyler, Manassein, Leube, Uffelmann, and others, that it is the acid, and not the pepsin, of the gastric juice which suffers diminution in fever, and it suggests the more extensive use of hydrochloric acid during the period of elevated temperature.—*Med. Times and Gaz.*, Nov. 10, 1877.

Salicine in Rheumatism.

Mr. Samson Gemmell and Mr. Frank Shearer contribute a paper on this subject to the *Glasgow Medical Journal* (Oct. 1877), in which they give the following conclusions:—

1. In acute febrile articular rheumatism, free from all complications, salicylate of soda is the most powerful therapeutic means which we possess; it cures more rapidly than any other.
2. It is impossible to assign to the treatment a uniform duration of days.
3. It does not prevent the cardiac, pulmonary, and cerebral complications of acute rheumatism; and where these exist prior to the treatment, it has no effect on them.
4. In spite of its antipyretic properties, it does not hinder the ascent of the temperature, which announces the advent of visceral complications.
5. In acute rheumatism, with slight complications, it is well to push the salicylate for its antipyretic and analgesic effects, but the use of revulsives should not be forgotten.
6. In acute rheumatism, with grave complications, it is well not to rely on the salicylate alone, but also to have recourse to other medicines.

Etiology of Typhoid Fever.

M. Ch. Bouchard read before the International Medical Congress, at Geneva, Sept. 12th, 1877, an interesting report on this subject, which is published in *Revue Mensuelle de Méd. et de Chir.* for Nov. The following are his conclusions:—

In the etiology of typhoid fever the doctrine of contagion and that of infection are both too exclusive. The doctrine of its fecal origin does not correspond with the universality of the facts. The doctrine of spontaneous development is not proved. Typhoid fever is a specific, miasmatic disease.

In its production it so happens that the morbific matter coming, we know not whence, but not necessarily from an infected organism, is capable of developing itself in animal matters which become then a focus of infection, and in the living human organism a focus of contagion. The contagion is almost always mediate.

The morbific matter arising from foci of infection, or of contaminated individuals, may contaminate the air, the soil, or the water. This morbific matter may be disseminated by men, by the contaminated objects, by the air, and especially by drinking water. The influence of reservoirs and of defective sewers, neglected or badly constructed, is shown, whether regarded as agents of infection or of mediate contagion. The influence of variations of the level of the subterraneous sheet of water is not clearly established.

Diphtheria complicating Enteric Fever.

At a late meeting of the Pathological Society of London (*Lancet*, Nov. 10, 1877), Dr. **GREENFIELD** showed a recent specimen of diphtheritic false membrane in the larynx and pharynx from a case of enteric fever. The case was that of a child, five years of age, who had been in St. Thomas's Hospital under the care of Dr. Murchison for about fifteen days. There was swelling and slight ulceration of Peyer's patches and solitary glands at the lower part of the ileum, the ulcers being small, round, and sharply excavated. In the larynx, upper part of trachea, and posterior surface of the palate and fauces, there was a thin layer of false membrane and muco-purulent secretion. The exudation was met with about the posterior nares and the orifices of the Eustachian tubes; but there was none on the anterior surface of the palate. Dr. Greenfield remarked that ulceration of the small intestines had been described in diphtheria, and he had often seen swelling of the solitary glands of the ileum in that disease; but the present was undoubtedly a case of enteric fever, and the questions raised by the case were, first, what was its relation to true diphtheria, and next whether diphtheria exists as a distinct specific disease at all.

Dr. **MURCHISON** said the patient in question was under his care at the hospital. The case was one of enteric fever in about the third week. Another child in the same family had recently had an abortive attack of enteric fever. In the present case laryngeal symptoms came on two days before death. No membrane could be seen on the fauces, which, with the tonsils, were red and swollen; and he hesitated to say whether the case was one of diphtheria complicating typhoid, or one of the other laryngeal complications that may occur in that disease. In addition to the laryngeal symptoms, there were signs of consolidation of the lungs. A form of diphtheria which he was in the habit of regarding as different from true diphtheria was not an unusual complication of enteric fever, and more often of typhus and scarlet fevers. Such cases occurred at the Fever Hospital, where it was curious that very few cases of diphtheria itself were admitted.

Dr. F. **SÉMON** had seen, both here and in Germany, cases of diphtheria complicating typhoid; and, as a rule, in such cases, the diphtheritic inflammation begins in the larynx and spreads upwards, but does not affect the upper part of the pharynx.

Dr. **MURCHISON** added that even when the laryngeal symptoms were severe, there had been no difficulty in swallowing. One of the members of the family had died of enteric fever. In reply to Mr. Pugin Thornton, he said that as a rule the complication occurred about the third or fourth week of the fever.